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Via Federal Express

Document Processing Center (Mail Code 7407M)
Room 6428
Attention: 8(e) Coordinator
Office of Pollution Prevention and Toxics
U.S. Environmental Protection Agency
1201 Constitution Ave., NW
Washington, DC 20460

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COMPANY SANITIZED

Dear 8(e) Coordinator:

Polyfluorosulfonic Acid

This letter is to inform you of the results of two genotoxicity tests with the test substance referenced above.

In Vitro Mammalian Chromosome Aberration test using Chinese Hamster Ovary Cells:

The test substance, was evaluated for genotoxicity in the *in vitro* mammalian chromosome aberration assay using Chinese hamster ovary (CHO) cells in both the absence and presence of an exogenous metabolic activation system (Aroclor-induced rat liver S9). The chromosome aberration assay was used to evaluate the clastogenic (chromosome breaking) potential of the test substance. A total of three test conditions were applied; 4-hour and 20-hour exposures without S9 metabolic activation, and a 4-hour exposure with S9 metabolic activation. Water was used as the negative control and test substance vehicle.

The highest dose level included in the study was 4280 µg/ml (equivalent to a 10 mM limit dose). The test substance was supplied as a 30% solution in water. Therefore, the dosing solution concentrations were adjusted using a correction factor of 3.33. Visible precipitate was observed in the treatment medium at dose levels $\geq$ 1284 µg/ml, and dose levels $\leq$ 428 µg/ml were soluble in the treatment medium. Substantial cytotoxicity (i.e., at least 50% cell growth inhibition relative to the solvent control) was observed at dose levels $\geq$ 428 µg/ml in all three test conditions. Both pH and osmolality where within normal ranges.

Based on these findings, the doses chosen for the chromosome aberration assay ranged from 12.5 to 300 µg/mL for all three treatment groups. Substantial cytotoxicity (i.e., at least 50% cell growth inhibition relative to the solvent control) was observed at 300 µg/ml in the 4-hour non-activated (74% reduction) and S9 activated (52% reduction) exposure groups, and at dose levels $\geq$ 250 µg/ml in the 20-hour non-activated (54% reduction) exposure group. Cytogenetic evaluations were conducted at 50 to 300 µg/mL (0.68 mM) for the 4-hour non-activated and activated exposure groups and at 50 to 250 µg/ml (0.58 mM) for the 20-hour non-activated exposure group.

The test substance was positive for the induction of structural chromosome aberrations in the presence and absence of S9 metabolic activation. The observed changes were statistically significant and dose dependent, and are considered biologically significant. The percentage of cells with structural aberrations in the 4-hour non-activated test condition was 23%. The percentage of cell in the corresponding positive control group (0.2 µg/ml mitomycin C) was 21%. The percentage of cells with structural aberrations in the 4-hour activated test condition was 21%. The percentage of cell in the corresponding positive control group (10 µg/mL cyclophosphamide) was 18%. The test substance was considered negative for the induction of structural aberrations in the 20-hour non-activated test condition. The compound was also considered negative for the induction of numerical aberrations in all three test conditions.

In Vivo Unscheduled DNA Synthesis In Primary Cultures of Rat Hepatocytes:

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The test substance was evaluated for unscheduled DNA synthesis in primary cultures of hepatocytes obtained from male Sprague-Dawley rats. The test substance, and the control substances were administered once by oral dose (gavage), and animals were sacrificed 2-4 hours and 12-16 hours after the treatment. The test substance was delivered in water. Concurrent negative (vehicle) and positive (dimethylnitrosamine, DMN) controls were included at both sacrifice time points.

A pilot toxicity assay was conducted to determine dose levels for the UDS test. In the pilot study, the test substance was administered via oral gavage to male rats at 1.0, 10, 100, 1000 and 2000 mg/kg body weight (bw). The high dose group consisted of 5 animals; the other dose groups consisted of 2 animals each.

All animals treated with 1.0, 10, 100 and 1000 mg/kg BW test substance appeared normal less than four hours following dosing, and 1, 2 and 3 days following dosing. All animals that were treated with 2000 mg/kg test substance appeared normal less than four hours following dosing. On day 1 following dose administration, 5/5 animals treated with 2000 mg/kg bw test substance appeared lethargic with 3/5 animals also appearing to have crusty noses and 1/5 animal also observed to have crusty eyes. On day 2 following dose administration 1/5 animal treated with 2000 mg/kg was found dead while the remaining 4/5 animals were observed to be normal. All remaining 2000 mg/kg animals were observed to be normal on Day 3 following dose administration. Significant body weight losses were observed for all animals treated with 1000 or 2000 mg/kg bw.

Based on the toxicity test, dose levels of 500, 1000, and 2000 mg/kg BW were administered in the UDS test to groups of male rats, with three rats per dose group evaluated in the test. No clinical signs or significant body weight changes were observed. The UDS test with mammalian cells *in vivo* was negative.

Under these experimental conditions, the findings described above appear to be reportable, based upon EPA's TSCA Section 8(e) reporting criteria.

Sincerely,